				Growth condition			
Locus	Gene	Protein Description	alleles	Ciprofloxacin	Naladixic acid	Trimethoprim	Conserved association?
FTN_0122	recA	Recombinase	3 of 3				+
FTN_0189	priA	Primase	3 of 3				
FTN_0399	radA	DNA repair	3 of 3				+
FTN_0412	recN	DNA repair	4 of 4				+
FTN_0632	dgt	dGTP triphosphatase	3 of 5				+
FTN_0787	rep	Helicase	5 of 5				
FTN_0891	ruvB	Helicase	4 of 4				
FTN_1025	ruvA	Helicase	2 of 2				+
FTN_1027*	ruvC	DNAse	2 of 2				+
FTN_1168*	xseA	Exonuclease	3 of 3				+
FTN_1177*	sbcB	Exonuclease	4 of 4				
FTN_1356	recD	Exonuclease	3 of 3				
FTN_1357	recB	Exonuclease	5 of 5				+
FTN_1359*	recC	Exonuclease	3 of 3				+
FTN_1513	xerC	Recombinase	3 of 3				+
FTN_1558	xerD	Recombinase	3 of 3				+

 \log_2 growth: -8.0

Figure S4. Quinolone antibiotic hypersensitive mutants. Mutations leading to increased ciprofloxacin and/or naladixic acid sensitivity are shown. Shadings reflect average growth of the two alleles with the strongest defects for each gene. Nutritional phenotypes are not shown. Asterisks indicate cases in which polar effects on expression of downstream genes could contribute to phenotypes. Several genes with strong mutant phenotypes that could be explained by polarity were not included in the table (FTN_0120, FTN_0121, FTN_0188, FTN_400, and FTN_401). The number of mutant alleles for each gene leading to increased sensitivity is provided. Functions associated with quinolone intrinsic resistance in *E. coli* are indicated.